Oxygen Breathing Accelerates Decompression from Saturation at 40 msw in 70-kg Swine

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Introduction: Submarine disaster survivors can be transferred from a disabled submarine at a pressure of 40 meters of seawater (msw) to a new rescue vehicle; however, they face an inherently risky surface interval before recompression and an enormous decompression obligation due to a high likelihood of saturation. The goal was to design a safe decompression protocol using oxygen breathing and a trial-and-error methodology. We hypothesized that depth, timing, and duration of oxygen breathing during decompression from saturation play a role to mitigate decompression outcomes. ***Methods:*** Yorkshire swine (67–75 kg), compressed to 40 msw for 22 h, underwent one of three accelerated decompression profiles: 1) 13.3 h staged air decompression to 18 msw, followed by 1 h oxygen breathing, then dropout; 2) direct decompression to 18 msw followed by 1 h oxygen breathing then dropout; and 3) 1 h oxygen prebreathe at 40 msw followed by 1 h mixed gas breathing at 26 msw, 1 h oxygen breathing at 18 msw, and 1 h ascent breathing oxygen. Animals underwent 2-h observation for signs of DCS. ***Results:*** Profile 1 (14.3 h total) resulted in no deaths, no Type II DCS, and 20% Type I DCS. Profile 2 (2.1 h total) resulted in 13% death, 50% Type II DCS, and 75% Type I DCS. Profile 3 (4.5 h total) resulted in 14% death, 21% Type II DCS, and 57% Type I DCS. No oxygen associated seizures occurred. ***Discussion:*** Profile 1 performed best, shortening decompression with no death or severe DCS, yet it may still exceed emergency operational utility in an actual submarine rescue.

Keywords: decompression illness, decompression sickness, staged decompression, isobaric oxygenation, disabled submarine.

IF THE HULL IS breached in a disabled submarine (DISSUB), internal pressure may rise and approach ambient sea pressure. This would increase nitrogen (N_2) partial pressure both in the remaining atmosphere and survivors' blood and tissues. Sailors in a DISSUB trapped at 40 meters of sea water (msw) with N_2 saturation face an estimated 80% probability of decompression sickness (DCS) if a direct ascent (dropout) to the surface is attempted (14,18). The NOAA air/oxygen decompression table for air/normoxic saturation predicts >57 h of air decompression time is required to safely surface from 40 msw (8). As rescue vehicle battery life and oxygen supplies are limited and deteriorating environmental conditions in the DISSUB may preclude a lengthy wait before decompressing each wave of survivors (10), this onerous decompression requirement in the face of such urgencies underscores the need for a shorter decompression schedule.

The new Submarine Rescue Diving and Recompression System includes the Submarine Decompression Chamber (SDC), a hyperbaric chamber capable of accommodating up to 32 rescued personnel and 4 tenders; and the Pressurized Rescue Module System (PRM), a rescue vehicle that can transport 16 survivors under pressures up to 5 atmospheres absolute (ATA) (~40 msw). Unfortunately, the PRM cannot directly transfer survivors to the SDC while maintaining pressure; furthermore, the SDC's capacity cannot accommodate treatment for an entire DISSUB crew. Performance estimates conclude the PRM can ascend from 610 msw to surface and depressurize from 40 msw to surface in 2 h for each 16 survivors. Therefore, rescue of a 155-man DISSUB crew, already a lengthy process based on these capabilities, would probably fail if a 57-h decompression schedule was required (16).

This leaves three possible scenarios for decompression of survivors in an emergency. One option is surface decompression on oxygen (Sur-D O_2), where survivors are quickly decompressed to the surface, transferred from the PRM to the SDC and recompressed to 40 msw, followed by a slow ascent breathing hyperbaric oxygen. Sur-D O_2 requires a 7-min surface interval between decompression and recompression. Because a DISSUB rescue will essentially be a saturation dive profile coupled with casualties needing medical assistance, the 7-min surface interval will probably be exceeded, increasing risk and requiring alternate approaches to reduce DCS risk.

A second option for reducing DCS risk is that of breathing oxygen at saturation depth on the DISSUB prior to decompression. This is known as "oxygen prebreathing" (OPB) and has been well studied. The efficacy of OPB to reduce DCS risk has been demonstrated in both swine and goats (5,12). In one 20-kg swine model, a 10-min OPB at 40 msw saturation followed by dropout decompression significantly decreased DCS incidence by 33% and delayed onset time from 11 min to 22 min compared with controls (6). In 70-kg swine, saturation at 18 msw followed by 1 or 2 h OPB prior to dropout decompression eliminated death and reduced Type II DCS

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by 77–85% (15). More recently, OPB for 45, 15, and even 5 min after saturation at 18 msw significantly reduced both Type I and II DCS with 45 min of OPB eliminating Type II DCS completely (11). In humans, OPB at 18 msw (2.52 ATA) saturation significantly reduced DCS, allowing for a safe 8–10-h decompression schedule (9). Unfortunately, it is unlikely that a DISSUB would have a pre-existing OPB capability; transport of oxygen to the DISSUB will be difficult and application of OPB at 40 msw depth might result in severe toxicities.

A third option, which we selected to focus on, is to use the PRM as a decompression vessel. As the PRM can decompress in 2 h, if a safe rapid decompression could simultaneously occur while transferring survivors to the surface, the incidence and severity of DCS might be mitigated. This could reduce DCS casualties, eliminate the need for treating an entire crew, and lower the likelihood of SDC capacity being overwhelmed. Similarly, delayed DCS onset might allow for more time for additional chamber assets to be flown in or for survivors to be evacuated to nearby chambers for recompression.

Although this proposed mechanism has since been refuted, in 1967 Behnke first hypothesized the benefits of oxygen breathing (OB) during decompression (2). OB has the potential for both CNS and pulmonary toxicity, however, in the 1970s Berghage described an "envelope" based on partial pressure and time constraints that balances oxygen's benefits with its potential toxicities in both OB and mixed gas decompressions (3,4). This suggests that OB while ascending or decompressing in the PRM, prior to recompression in the SDC, might be a feasible approach to shorten decompression time from >57 h to a more operationally suitable time. Here we report the results of three accelerated decompression profiles from a simulated DISSUB scenario at 40 msw. Using a simple trial-and-error methodology, we attempted an abbreviated decompression using air, OB, and OPB in various combinations in an attempt to reduce DCS incidence and severity, ideally to fit within the 2-h PRM decompression window.

METHODS

The animal experiments reported here were conducted according to the principles set forth by the National Research Council (13). Before commencing, our Institutional Animal Care and Use Committee reviewed and approved all aspects of this protocol. The institutional animal care facility is fully AAALAC accredited. All animals were maintained under the surveillance of veterinary staff.

Subjects

Neutered male Yorkshire swine (*Sus scrofa*, $N = 32$, 70.8 ± 3.8 kg) were examined by a veterinarian upon receipt. Subjects were housed individually in free running cages with full access to water, food (2% of bodyweight daily, Lab Porcine Diet Grower 5084, PMI Nutrition, Brentwood, MO) and environmental enrichment for 5 d of acclimatization prior to any procedures.

Pre-Dive Preparation

On the day prior to hyperbaric exposure, subjects were moved to a surgical suite for external jugular vein catheter placement. Anesthesia induction was performed with intramuscular injection of 20 mg · kg^{-1} ketamine and 2 mg · kg^{-1} xylazine (Ketaject 100 mg · ml^{-1}, Xyla-Ject 100 mg · ml^{-1}, respectively; Phoenix Pharmaceutical, St. Joseph, MO). Anesthesia was maintained with 2–5% isoflurane (Halocarbon Products, Rover Edge, NJ) via a face mask. The external jugular vein was catheterized with a 16-gauge by 20.3-cm single lumen catheter (Braun Certofix; B. Braun Medical Inc., Bethlehem, PA) via the modified Seldinger technique (1) and advanced until 8-10 cm extended from the skin incision site. The catheter was sutured in place with an exit site on the dorsal thorax, taped to the skin, and then brought through a vest worn by the animal, thus securing and protecting the catheter line and injection port. The vest was designed to accommodate a 76-cm long, 8-cm diameter Tygon™ tube (Cole-Parmer, Vernon Hills, IL) to be attached to the catheter on the day of the dive. This allowed administration of medication or fluids while the animal was inside the chamber under pressure. Limited ambulation was assessed in the box during the observation period; full ambulation after recovery was verified prior to return to the holding pen, where the animal remained overnight.

On the day of the hyperbaric exposure, the subjects were placed into individual custom-designed Plexiglas™ boxes (26 x 54 x 38) inside a steel-hulled hyperbaric chamber reported elsewhere (11). Each box allowed for a hyperbaric oxygen environment in which the subjects could breathe without requiring restraints. Subjects had access to water ad libitum via a lixor fitted to the boxes. The external jugular vein catheter was connected to a sterile line and fed through a Tygon™ tube secured to the torso vest and a 360° swivel on the ceiling of the Plexiglas box, which allowed the animal to move freely and to make postural adjustments without twisting the line. The sterile line was passed through a hull penetrator port of the chamber and connected to a high-pressure positive displacement infusion pump (Mini pump; Milton Roy, Ivyland, PA), allowing for intravascular infusions or blood withdrawals while under pressure.

Hyperbaric Exposure

The chamber was pressurized with air to 40 msw (5 ATA) at a rate of 9.1 m · min^{-1}. Subjects were monitored via close circuit television for any signs of distress related to middle ear barotrauma. The subjects remained at 40 msw for 22 h, a period accepted to be sufficient for inert gas saturation in 20-kg swine (7) and considered sufficient in 70-kg swine. Water was provided ad libitum and the animal remained unrestrained within the Plexiglas box throughout the dive. Chamber and box atmospheres were monitored with separate gas analyzers (Geotech Anagas Dive Analyzer, Denver, CO). The chamber oxygen concentration was maintained at 21% (± 0.02%) and CO_2 at < 0.05% surface equivalent. The

oxygen concentration in the Plexiglas box was adjusted according to the dive profile to allow either air (21% O_2) or OB (32–95% O_2). Changes in box atmosphere were achieved by flushing the box with oxygen, mixed gas, or air. Changes in the breathing gas composition of the Plexiglas boxes (e.g., from 95% O_2 to air) were accomplished in about 5 min. Temperature was maintained between 75–85°F (23.9-29.4°C) with 50% (± 5%) humidity via an environmental control. After 22 h at 40 msw, paired subjects sequentially underwent one of the following decompression profiles (**Table I**):

Profile 1—staged decompression: Pigs were decompressed according to an abbreviated air decompression schedule from 40 msw to 18 msw over a 13-h, 16-min duration. The decompression rate between stops was 9.1 m · min^{-1}. At the 18-msw stop the breathing gas was switched to ~95% (2.66 ATA) O_2 for 1 h. The subjects were then decompressed directly to the surface at 9.1 m · min^{-1} while still breathing 95% O_2.

Profile 2—rapid decompression: Subjects were brought directly from 40 msw to 18 msw on air at 9.1 m · min^{-1}. At the 18-msw decompression stop, subjects breathed 2.66 ATA (95%) O_2 for 1 h, followed by decompression to the surface at 9.1 m · min^{-1} while breathing 95% O_2.

Profile 3—rapid decompression using mixed gases: The Plexiglas box atmosphere was changed to 32% O_2 (1.6 ATA) 1 h before decompression. After breathing OPB for 1 h the Plexiglas box atmosphere was switched back to air and the pigs decompressed at 1.5 m · min^{-1} to 26 msw. At 26 msw, the oxygen fraction was increased to 50% (1.79 ATA) and subjects breathed this mixture for 1 h. Subjects were then switched back to air and brought to 18 msw at 0.3 m · min^{-1}. The pigs were held at 18 msw for 1 h while breathing 95% O_2 (2.66 ATA). Following a 15-min air break at 18 msw, the pigs were decompressed to the surface at 0.3 m · min^{-1} on 95% O_2.

Post-Dive Observation

For the three profiles tested, the breathing mixture was switched to air upon reaching the surface. Observers entered the chamber to record any signs of DCS while the pigs remained inside their Plexiglas containers for a 2-h observation period. Observations were recorded at ≤ 10-min intervals until subjects became severely symptomatic and were euthanized, spontaneous death occurred, or the 2-h observation period was completed. A 2-h post-dive observation period has been determined sufficient to detect all symptoms of severe DCS in a swine saturation model, as observed symptoms plateau at 1 h after surfacing (6). Heart rate and arterial oxygen saturation (S_aO_2) were monitored continuously using pulse oximeters (Heska, model #4404, Des Moines, IA).

Type I DCS included cutis marmorata, defined as observed cyanotic patches on the animal's skin, and pain-only DCS, defined as impaired limb movement (foot curling, limb lifting) without weakness or other neurological findings. Type II DCS included neurological and cardiopulmonary signs. Neurological DCS was defined as motor weakness (limb weakness, repeated inability to stand after being righted by the investigator), paralysis (complete limb dysfunction, areflexia, or hypotonia), or sensory compromise (e.g., failure to retract from painful stimuli). Cardiopulmonary DCS was defined as a visually observed respiratory rate > 60 breaths · min^{-1} combined with respiratory distress, as evidenced by open-mouthed, labored breathing, central cyanosis, or the production of frothy white sputum. The onset of severe DCS (neurological or cardiopulmonary dysfunction) and all other behavioral signs and symptoms were recorded to the nearest minute. Pigs with signs of severe DCS were given Diazepam (2.5 mg, intravenous, Hospira, Inc., Lake Forest, IL) through the in-dwelling catheter as necessary to alleviate distress. If signs of imminent death were evident or their distress was not relieved with Diazepam, the animal was euthanized with pentobarbital and phenytoin (Euthasol® 1 cc/10 lb bodyweight i.v., DelMarva Laboratories, Inc., Midlothian, VA). After the 2-h observation period, surviving subjects were removed from the chamber and examined for signs of neurologic, cutaneous, or cardiopulmonary DCS. They were then placed into holding pens for an additional 22 h. After 24 h, surviving animals were euthanized.

Selection of Dive Profile

We employed a trial-and-error design that has been previously described (17). A priori accept/reject criteria were selected to expedite examination of dive profiles we believed to yield reasonable risk levels in a DISSUB contingency situation. The selected risk limits were: ≤ 10% mortality (either death or severe DCS requiring euthanasia), ≤ 20% severe DCS, and ≤ 30% oxygen toxicity

TABLE I. DECOMPRESSION PROFILES FOR THE EXPERIMENTS.

Profile 1							
Decompression stop depth [msw (fsw)]	40 (132)	26 (85)	24 (80)	23 (75)	21 (70)	20 (65)	18 (60)
Time at stop (h:min)	22:00	2:28	2:33	2:39	2:45	2:51	1:00
Percent oxygen concentration	21	21	21	21	21	21	95
Profile 2							
Decompression stop depth [msw (fsw)]	40 (132)	18 (60)					
Time at stop (h:min)	22:00	1:00					
Percent oxygen concentration	21	95					
Profile 3							
Decompression stop depth [msw (fsw)]	40 (132)	40 (132)	26 (85)	18 (60)			
Time (h:min)	22:00	1:00	1:00	1:00			
Percent oxygen concentration	21	32	50	95			

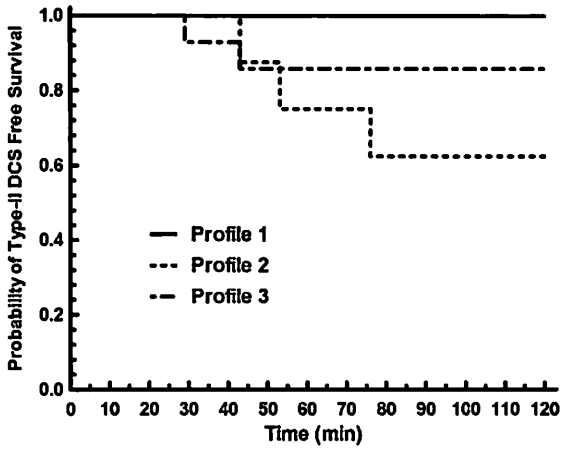

Fig. 1. Kaplan-Meier curves for the probability of Type II decompression sickness (DCS) free survival vs. time.

during the 2-h post-dive observation period. The intent was to take the original safe NOAA 57-h decompression, design a significantly shortened profile, and test the new decompression profile. If the profile was found to be "reasonably safe" based on the risk limits described, the next decompression profile to be tested would be shortened further. If the profile proved "very risky" (exceeded risk limits), then it would be modified until it met criteria as "reasonably safe"; each subsequent profile would be tested in the area between the most recently tested "reasonably safe" and "very risky" profiles. The maximum number of subjects to dive on any 1 profile was 15, but as soon as a reject threshold was achieved, testing was discontinued for that profile. With 15 dives, this rule meant that we would accept as "reasonably safe" 3 cases of cardiopulmonary or neurologic DCS or < 2 subjects with Type II DCS severe enough to warrant euthanasia. We began with dive Profile 1, a modified version of a standard staged air decompression, accelerated with oxygen at 18 msw.

Statistical Analysis

Analyses were conducted using SAS (SAS Institute Inc., Cary, NC). Statistical significance was established at $P < 0.05$. Primary endpoints were rates of death (euthanasia), Type I (cutis or pain), and Type II (cardiopulmonary or neurological) DCS during the 2 h after surfacing. The secondary endpoint was the rate of Type II DCS during the 24-h observation period. Primary and secondary endpoints were assessed using Fisher's exact test. Type II DCS event-free survival during the 2-h observation period was assessed using Kaplan-Meier methodology; differences were compared using the log-rank test. *P*-values were not adjusted for multiplicity.

RESULTS

We have previously demonstrated that "dropout decompression" (direct ascent to the surface from 22-h saturation at 18 msw) in a 15-swine cohort (mean weight 69.5 kg) resulted in 86.6% Type I cutis, 40% cardiopulmonary DCS, and 73.3% neurologic DCS (11). The anticipated occurrence of DCS in a dropout from 40 msw was anticipated to equal or exceed these rates. During the initial 2-h observation period, there was no significant difference between dive profiles for required euthanasia. Swine on Profile 1 had a significantly lower incidence of Type I cutis compared with Profiles 2 and 3 ($P < 0.01$). Type I pain incidence was not statistically significantly different between groups. Similarly, Type II DCS incidence was not statistically significantly lower in Profile 1 compared to Profile 2 or 3 ($P = 0.10$). All of the above were generated using Fisher's Exact Test. Time to Type II DCS onset during the 2-h observation is represented by Kaplan-Meier curves in **Fig. 1** ($P = 0.12$).

DCS incidence is summarized in **Table II** while onset times are indicated in **Table III** for the three profiles. Profile 1 resulted in no cases of severe (Type II) DCS during the 2-h observation period and only two cases (20%) of Type I DCS (pain only). In Profile 2, 3/8 (38%) of the subjects exhibited signs of severe DCS during the 2-h observation period (one neurological, one cardiopulmonary, and one both), two (25%) of which required euthanasia within the 24-h follow-up period due to severe cardiopulmonary DCS. A fourth animal experienced neurologic DCS after 2 h, 36 min. No pig manifested pain, but 6/8 (75%) experienced cutis, ranging from 31–112 min after surfacing. Of the 14 animals in Profile 3, 2 (14%) suffered severe DCS during the 2-h observation period and required euthanasia; a 3rd had neurologic signs at 2 h, 28 min. Two (14%) manifested signs of pain (one with accompanying cutis) and seven (50%) experienced cutis 5–165 min after surfacing.

On average, the swine in decompression Profile 1 were significantly heavier than those in Profile 2 or 3 ($P < 0.05$) (Table II). Although we were not able to determine whether any animals experienced pulmonary oxygen toxicity at depth, none exhibited symptoms of oxygen toxicity, such as seizures or tachypnea, during OB or OPB. Some swine elected not to ambulate in the Plexiglas boxes upon surfacing and during the 2-h observation period. As a result two cases of neurologic DCS and one of cutis were not detected until a full examination was performed in the animal run facility. However, we believe that these signs probably developed within the 2-h observation period and that these late findings reflect postural limitations to the observation rather than delayed onset of DCS.

DISCUSSION

In this study we demonstrated that Profile 1, which used five air stops from 40 msw saturation to 18 msw (over a period of 13.27 h), coupled with 1 h OB at 18 msw then 'drop-out' completely prevented spontaneous death and severe DCS warranting euthanasia. Although this was not statistically superior to the other two profiles for the primary endpoint, this is probably due to the small numbers and low death rates experienced in those profiles as well. While *P* approached significance at 0.1, because we did not adjust for multiplicity, this interpretation requires caution. Certainly the de-

TABLE II. OUTCOME OF DECOMPRESSION SCHEDULE IN THE THREE PROFILES EVALUATED (N = 32).

	Dive Profile 1 (N = 10)	Dive Profile 2 (N = 8)	Dive Profile 3 (N = 14)
Weight (kg), mean ± SD.	73.6 ± 3.9	70.3 ± 2.6	69.7 ± 3.8
During 2-h follow-up period (Primary)			
Death, N (%)	0	2 (25)	2 (14)
Type II DCS, N (%)	0	3 (38)	2 (14)
Cardiopulmonary	0	2 (25)	2 (14)
Neurologic	0	2 (25)	1 (7)
Type I DCS, N (%)	2 (20)	6 (75)	7 (50)
Pain	2 (20)	0	2 (14)
Cutis	0	6 (75)	6 (43)
During 24-h follow-up period (Secondary)			
Death, N (%)	0	1 (13)	–
Type II DCS, N (%)	0	1 (13)	1 (7)
Cardiopulmonary	0	–	–
Neurologic	0	1 (13)	1 (7)
Type I DCS, N (%)	–	–	1 (7)
Pain	–	0	–
Cutis	0	–	1 (7)

– = No further signs noted after 2 h.

crease in Type II DCS and death from one or two to zero cases is of clinical importance despite not being statistically significant. We note OB for 1 h at 18 msw allowed for drop-out, eliminating an expected 20–24-h decompression obligation from 18 msw to the surface. A total decompression time of approximately 14.3 h was achieved with no incidence of cardiopulmonary or neurologic DCS and only 20% Type I DCS in the 24-h observation period. We observed statistically lower rates of Type I DCS in Profile 1 than the other two profiles and the incidence of DCS among the heavier subjects of Profile 1 was lower. These findings suggest Profile 1 was safer and that weight was not a confounding factor in DCS outcome probabilities.

While we accept this profile as relatively safe, it is probably not operationally useful as a decompression strategy in the PRM due to the long turn-around times for each evacuation, particularly if a full crew of 155 survivors must be rescued from a flooding DISSUB rapidly losing breathable air. Using our trial-and-error methodology, the next most applicable profile would be one to match the shortest possible (2 h) transit and decompression time capabilities of the PRM. Since Profile 1 succeeded using OB at 18 msw and 1-h OPB prevented death after dropout from 18 msw saturation (11,12,15), we attempted Profile 2, a direct ascent from 40 msw to 18 msw followed by 1-h OB and dropout. Profile 2 did not demonstrate this approach to be safe, resulting in 75% Type I DCS, 50% Type II DCS, and 25% death from euthanasia. Having exceeded our risk thresholds, Profile 2 should be rejected as "unsafe" as an emergency decompression profile. We also exposed two animals to a dive using 2 h of OB at 18 msw, with one case (50%) of severe DCS requiring euthanasia. We then conceded that a direct ascent from 40 to 18 msw followed by oxygen decompression and dropout strategy is futile (Mahon RT; unpublished results; 2006). Profile 2 probably exceeds the ability of the OB to prevent DCS; extending the OB period at this depth appeared to add no benefit. Our results from Profiles 1 and 2, plus previous OBP saturation

TABLE III. TIME TO ONSET OF SYMPTOMS FOR TYPE I (CUTIS) AND TYPE II (CARDIOPULMONARY AND NEUROLOGIC) DCS BY PROFILE.

		Time of DCS Onset (min)*		
Subject Number	Profile number	Cutis	Cardiopulmonary DCS	Neurologic DCS DCS
12990	2	112	-	-
12989	2	50	-	-
13428	2	57	76	75
13429	2	57	-	156
13602	2	31	43	-
13603	2	52	-	53
17808	3	165	-	-
18082	3	26	29	26
18022	3	5	-	-
18168	3	40	43	-
18169	3	29	-	-
18508	3	81	-	-
18632	3	54	-	-
18631	3	-	-	148

* Onset of exact time of Type I pain-only signs was too difficult to assess.

studies (11,12,15) at 18 msw suggest OB at 18 msw is most effective at preventing DCS for the last 18 m of the ascent, but not the deeper ascent to the 18 msw stop.

Using our trial-and error method, we noted that OB success accelerating ascent from saturation at 18 msw was not completely related to its duration. We hypothesized that timing, with more frequent periods of OB, might be more effective at reducing DCS rates and OB might also be employed at deeper stops to reduce the 13.3 h decompression required from 40 msw to 18 msw that was safely used in Profile 1. Furthermore, more information is needed about the safety of OB in combination with OPB in the event an accelerated profile is derived for the PRM and survivors do an OPB before escaping. A previous study showed OPB for as little as 1 h prior to dropout from 18 msw saturation completely prevented death in 70-kg swine (15). Latson was unable to show that OB at decompression stops < 18 msw decreased DCS in humans decompressing from 18 msw saturation until OPB at 18 msw was added to the decompression schedule (9). We reproduced this effect in Profile 3 with OPB at 40 msw, on the hypothesis that initiating oxygen at the deepest portion of the profile would accelerate the initial ascent phase. Unfortunately, OPB benefits might be lost while subjects breathed air during ascent to 18 msw if tissue beds that take up and release inert gas quickly (fast compartments) reaccumulated inert gas. We hypothesized that adding an OB period at 26 msw in Profile 3 would prevent some of this phenomenon.

Pure (100% O_2) OB at 40 msw (5 ATA) or at 26 msw (3.6 ATA) would present a high risk for oxygen toxicity, so for Profile 3 we selected gas mixtures with an oxygen content protective against DCS, but hypothetically low enough not to elicit oxygen seizures. Berghage and McCracken demonstrated the optimal partial pressure for 1 h of oxygen at 10 ATA ambient pressure is about 2.7 ATA in rats, and suggested shallower depths have a higher oxygen concentration "safe envelope" (3). We chose 1.6–1.8 ATA O_2 to minimize seizure risk. While mixed gases present technical challenges, a highly effective mix would justify efforts to overcome the technical hurdles for DISSUB operations. We further attempted to shorten decompression time in Profile 3 by adding a 1-h ascent from 18 msw to the surface with OB (total of 4 h of oxygen) instead of a 'drop-out' to the surface as in Profiles 1 and 2.

While 32% O_2 OPB at 40 msw did not cause seizures, Profile 3 ultimately resulted in 57% Type I DCS, 21% Type II DCS, and 14% requiring euthanasia. Furthermore, Profile 3 did not result in statistically superior outcomes for DCS incidence or euthanasia compared to Profile 2. Higher partial pressures such as 44% O_2 (Berghage's 2.7 ATA maximum partial pressure) at 5 ATA might yield better results and even be the focus of future efforts. However, seizure risk will also be elevated and delivering two mixed gases on a DISSUB and rescue vehicle is beyond the current (and foreseeable) capabilities of the PRM. Since the animals breathed air following OPB completion and during ascent to the initial HBO decompression stop (40–26 msw), the higher DCS incidence of Profile 3 may represent fast compartment nitrogen reaccumulation during ascent. If this is the case, even a higher partial pressure of oxygen with attendant increased seizure risks might not be sufficient to overcome this phenomenon. Due to these multiple technical challenges, limitations of rescue equipment to mix gases, and results > 20% severe DCS or 10% death/euthanasia, we reject Profile 3 as very risky. Ultimately, a profile between 4 and 14 h of OB will be the most feasible for rapid decompression from 5 ATA. Using residual nitrogen time modeling to determine where to add the oxygen stops, deeper or shallower than 18 msw, would ideally optimize a profile.

Our study does have some limitations. Caution is urged given the small numbers of animals studied and the difficulty of determining neurologic deficits in unsedated swine. This study was designed for use in the PRM; as such it probably cannot be interpreted for use in the SDC because our animals probably began the decompression with no bubbles in their system. It is reasonable to assume survivors of a PRM accelerated decompression with OB or an OPB will have bubbles present in their body prior to recompression and decompression due to the surface interval/lack of transfer under pressure capability. Bubbles present at the beginning of decompression grow much larger than those formed during decompression because they are present for the complete pressure change. Hence, an accelerated decompression profile for the SDC would require a different experimental model.

This study demonstrates that incorporating OB into an emergency decompression strategy from saturation at 40 msw can significantly accelerate total decompression time to 14.3 h (Profile 1) without compromising safety. This may be of benefit to a DISSUB scenario depending on depth, number of survivors, available time for rescue, and availability of OB on the PRM. However, 1–4 h of accelerated decompression with OB ± OPB, while reducing DCS incidence, is probably unsafe for emergency use in all but the direst circumstances. Continued modification(s) to this decompression schedule with more and varying oxygen periods merit further study.

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